susceptibility in adolescent boys, in whom the virus continues to circulate.[7]

Surveillance has shown that increasing numbers of cases of measles are already occurring in schools, confirming the probability of an epidemic if action is not taken promptly.[2] Late last year and early this year several health boards in the west of Scotland saw a large increase in measles—mainly in secondary schoolchildren—which resulted in 138 admissions to one infectious disease unit alone (P Christie, personal communication). If applied to England and Wales the incidence in the Scottish outbreak would give rise to the 100 000-200 000 cases predicted by the mathematical models. The five deaths among the 10 000 cases that occurred during a recent outbreak in schoolchildren in Quebec were further evidence of the severity of measles in older age groups and confirmed the mortality predictions of the modellers.[8]

The resurgence of measles in Britain is largely the result of poor vaccine coverage in the past, but over a tenth of cases occur in children who have been vaccinated.[2] Vaccine failure is known to sustain transmission in populations with high vaccine coverage,[9] and this is the rationale for including all schoolchildren in the campaign irrespective of whether they have been vaccinated against measles previously. The next logical step will be a recommendation of a two dose schedule—a strategy being adopted by an increasing number of countries which like Britain are seeking to eliminate measles.[9] Several countries that have achieved high coverage with a single dose have experienced epidemics after "honeymoon" periods of low incidence.[9] Such epidemics are attributable to an accumulation of susceptible subjects, both unvaccinated children and those in whom vaccination has failed, and are triggered when their number reaches a critical threshold. There is therefore a great epidemiological advantage in introducing a second dose in the wake of a campaign to maintain the number of susceptible subjects well below this threshold.[1] The simplest strategy would be to give a second dose at school entry—when boosters of the diphtheria-tetanus and oral polio vaccines are given—but whether this strategy would be successful in reaching the 7-8% of children who miss the first dose is unknown.[10] Periodic mass campaigns might be a more effective way of reaching this group, but relying on them without a guarantee that resources would be available when required would be risky. Speculation on whether this approach is a serious option in Britain must await the outcome of the present campaign.

The campaign approach for delivering measles vaccine has not been tried before in an industrialised country but has been used successfully in the Caribbean and Central and South America, where measles is now at an all time low.[11] In Cuba transmission of measles seems to have been interrupted after a massive campaign in 1989 that achieved over 95% coverage of children aged 1-14.[11] It will be interesting to see whether the immunisation services in Britain can match this performance. Whatever the operational outcome, the comprehensive surveillance systems in place (which now include routine salivary diagnosis in all suspected cases[2]) will allow the epidemiological consequences of the campaign to be assessed accurately.

ELIZABETH MILLER
Head

Immunisation Division,
PHLS Communicable Disease Surveillance Centre,
London NW9 5EQ

1 Babad HR, Nokes DJ, Gay NJ, Miller E, Morgan-Capner P, Anderson RM. Predicting the impact of measles vaccination in England and Wales: model validation and analysis of policy options. *Epidemiol Infect* (in press).
2 Ramsay M, Gay N, Miller E, Rush M, White J, Morgan-Capner P, *et al.* The epidemiology of measles in England and Wales: rationale for the national vaccination campaign. *CDR Review* (in press).
3 Wassilak SGF, Orenstein WA, Strickland PL, Butler CA, Bart KJ. Continuing measles transmission in students despite a school-based outbreak control program. *Am J Epidemiol* 1985;122:208-17.
4 Davis RM, Whitman ED, Orenstein WA, Preblud SR, Markowitz LE, Hinman AR. A persistent outbreak of measles despite appropriate prevention and control measures. *Am J Epidemiol* 1987;126:438-49.
5 Brown D, Ramsay M, Richards A, Miller E. Salivary diagnosis of measles: a study of notified cases in the United Kingdom, 1991-93. *BMJ* 1994;308:1015-7.
6 Morgan-Capner P, Wright J, Miller CL, Miller E. Surveillance of antibody to measles, and rubella by age. *BMJ* 1988;297:770-2.
7 Miller E, Tookey PA, Morgan-Capner P, Hesketh L, Brown DWG, Waight PA, *et al.* Rubella surveillance to June 1994: third joint report from the PHLS and the national congenital rubella surveillance programme. *CDR Review* (in press).
8 McLean ME, Carter AO. Measles in Canada - 1989. *Canada Diseases Weekly Report* 1990; 16-42:213-8.
9 Cutts F, Markowitz L. Successes and failures in measles control. *J Infect Dis* (in press).
10 White J, Leon S, Ramsay MEB. COVER (cover of vaccination evaluated rapidly) 31 *CDR Review* 1994;4:R129-30.
11 Measles elimination in the Americas. *Bulletin Pan Am Health Organisation* 1992;26:271-4.

The eradication of gonorrhoea

Can be achieved

The continued global survival of *Neisseria gonorrhoeae* is inevitable because of its immunobiology,[1] the seeming lack of natural or acquired immunity, human sexual behaviour, and the frequency of asymptomatic infection. Nevertheless, endemic gonorrhoea could be eradicated from Britain in the 1990s. Sweden has achieved this public health goal, and many other developed countries report dramatic reductions in incidence.[2] In Britain reported cases of gonorrhoea have declined every year since 1977, with a particularly big decrease in 1987. Many health districts have already achieved the nationally set target for 1995 of fewer than 49 cases per 100 000 population aged 15-64.[3] Geographical variation in the incidence of gonorrhoea in England is, however, considerable, with some urban areas still substantially above this target.[4]

Has any one factor been decisive in recent successes in controlling gonorrhoea? Certainly, radical innovations in treatment cannot take the credit. Effective drugs have been available throughout the rise and fall in the incidence of gonococcal infection during the 1960s, '70s, and '80s. Effective treatment is, however, a key component in control and has required some major changes in the past 30 years. Even before the emergence of penicillinase-producing *N gonorrhoeae* in 1976, increasing resistance to penicillin had been widely reported. The reduced sensitivity to penicillin was associated with failure of treatment but did not prove a major problem, as was once feared.[5] Higher doses of penicillin coupled with probenecid re-established high rates of clinical cure.

The appearance and subsequent rapid spread of penicillinase-producing *N gonorrhoeae* demanded an alternative strategy. In sharp contrast to the reported high prevalence of this strain in some parts of the world (notably Africa and South East Asia), its incidence in Britain has remained

low. Indeed, isolates of penicillinase-producing *N gonorrhoeae* as a proportion of all cases of gonorrhoea in Britain peaked at 2·3% in 1984 and have subsequently declined. Even in London, the penicillinase-producing strain accounts for less than 4% of gonoccocal infections, and many of these cases are imported.[6,7] As the incidence of gonorrhoea has fallen most genitourinary medicine departments have not reported a rising proportion of penicillinase-producing *N gonorrhoeae*. Only 36 isolates were reported by the Public Health Laboratory Service in England during the first quarter of this year.[8] Reports of non-penicillinase-producing chromosomally mediated penicillin resistant *N gonorrhoeae* appeared in the early 1980s. As with penicillinase-producing strains, this chromosomally mediated resistance to penicillin is distributed worldwide, has been associated with specific outbreaks of gonorrhoea, but is uncommon in Britain.[8]

Cephalosporins (notably ceftriaxone, cefoxitin, and cefotaxime) and spectinomycin have provided effective treatment where resistance to penicillin is common. Ceftriaxone has been widely used in North America and South East Asia for nearly a decade with no reduction in its clinical efficacy. Chromosomally mediated resistance to spectinomycin has been described after use of this antibiotic as first line treatment, but it remains a valuable treatment.[9] Fluoroquinolones became popular as a first line treatment for uncomplicated gonoccocal infection in the mid-1980s and are widely used in British genitourinary medicine clinics. They offer single dose oral treatment and are highly effective against penicillin resistant strains. They should not be used to treat pregnant women, children, or adolescents. Perhaps inevitably, the development of clinically important resistance to ciprofloxacin has recently been reported,[10] but resistance to fluoroquinolones is uncommon in Britain and does not seem to be increasing.[11] Most resistant isolates have been imported from Spain and the Far East, and most also carry plasmid or chromosomally mediated resistance to penicillin.

How should uncomplicated gonorrhoea be treated in Britain? Ampicillin or amoxycillin taken with probenecid remains effective as a single dose oral first line treatment. Fluoroquinclones are a popular alternative and have the advantage of efficacy against strains resistant to penicillin, but resistance to quinolones needs monitoring.[7] If the gonococcal infection has been contracted overseas, treatment with penicillin is inappropriate and a quinolone, cephalosporin, or spectinomycin should be used. Treatment for gonorrhoea should routinely be combined with effective treatment against *Chlamydia trachomatis* in recognition of the high coinfection rate and the high incidence of postgonococcal urethritis. Azithromycin offers the prospect of single dose oral treatment effective against both *N gonorrhoeae* and *C trachomatis*.[12] It is expensive; gastrointestinal side effects are common if the higher, 2 g dose is used; and resistance to macrolides has been observed with erythromycin. Whatever antibiotic is used, when gonorrhoea is diagnosed the bacteria should be isolated and antibiotic sensitivities monitored.

The treatment of gonorrhoea may have changed with the development of resistance to antibiotics, but its efficacy has been maintained, albeit at a price that is not affordable by all nations. Effective treatment is, however, only one element in the successful control of gonorrhoea. It needs to be backed up by follow up to ensure clinical cure, contact tracing, and education to influence behaviour and reduce the risk of patients acquiring further sexually transmitted infections. The epidemic of AIDS has undoubtedly given a major impetus to sex education in schools, increased public awareness about sexually transmitted diseases, and encouraged the modification of behaviour and the use of condoms. Britain has yet to embark on programmes of widespread screening for asymptomatic infection with *N gonorrhoeae* and *C trachomatis*, and the momentum of programmes to reduce risk in sexual behaviour needs to be maintained. The eradication of endemic gonorrhoea in Britain should become a goal, but its achievement will require a more proactive approach modelled on that in other countries.

CHRIS BIGNELL
Consultant in genitourinary medicine
Department of Genito-Urinary Medicine,
City Hospital,
Nottingham NG5 1PB

1 Cohen MS, Sparling PF. Mucosal infection with Neisseria gonorrhoeae. Bacterial adaptation and mucosal defenses. *J Clin Invest* 1992;**89**:1699-705.
2 Kihlstrom E, Danielsson D. Advances in biology, management and prevention of infections caused by Chlamydia trachomatis and Neisseria gonorrhoeae. *Current Opinion in Infectious Diseases* 1994;7:25-33.
3 Department of Health. *Health of the Nation: a strategy for health in England*. London: HMSO, 1992.
4 Blair I, Skett S. Gonorrhoea in the west Midlands. *Communicable Disease Report* 1994;4: R25-8.
5 Willcox RR. A survey of problems in the antibiotic treatment of gonorrhoea. *British Journal of Venereal Disease* 1970;**46**:217-42.
6 Sherrard J, Barlow D. PPNG at St Thomas' Hospital—a changing provenance. *International Journal of STD and AIDS* 1993;4:330-2.
7 Lewis DA, Forster GE, Hooi AY, Goh BT, Wisdom AR. *Gonococcal infection in east London: a two centre microbiological study*. Liverpool: Medical Society for the Study of Venereal Diseases Spring Meeting, 1994. (Abstract 85.)
8 Public Health Laboratory Service. Neisseria gonorrhoeae: laboratory reports of antibiotic resistant isolates, January to March 1994. *Communicable Disease Report* 1994;4:67.
9 Boslego JW, Tramont EC, Takafuji ET, Diniega BM, Mitchell BS, Small JW, *et al*. Effect of spectinomycin use on the prevalence of spectinomycin-resistant and of penicillinase-producing Neisseria gonorrhoeae. *N Engl J Med* 1987;317:272-7.
10 O'Mahony C, Timmins D. Treatment failure using double dose ciprofloxacin in a case of highly resistant gonorrhoea. *Genitourin Med* 1992;**68**:274-5.
11 Turner A, Gough KR, Jephcott AE. *Epidemiology of gonococci with reduced sensitivity to ciprofloxacin isolated in the UK*. Liverpool: Medical Society for the Study of Venereal Diseases Spring Meeting, 1994. (abstract 87.)
12 Handsfield HH, Dalu ZA, Martin DH, Douglas JM, McCarty JM, Schlossberg D, *et al*. Multicenter trial of single-dose azithromycin vs ceftriaxone in the treatment of uncomplicated gonorrhoea. *Sex Transm Dis* 1994;**21**:107-11.

Correction

Slowing the march of the Marlboro man

An author's error occurred in this editorial by Ronald M Davis (8 October, p 889-90). The references numbered 3 and 6 were transposed; therefore reference 3 in the text refers to the World Bank's 1993 report.